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Study the Role of Serum visfatin in Type 2 Diabetic Patients with and without Hemodialysis

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ABSTRACT

Background: The incidence and prevalence of diabetes mellitus have grown significantly throughout the world, due primarily to the increase in type 2 diabetes. Chronic kidney disease (CKD) is a frequent complication of type 2 diabetes mellitus (T_2DM), Visfatin has insulinomimetic effects by activating the insulin signal transduction pathway through binding to the same receptor. According our study have shown the relationship between visfatin a specific biomarker for T2DM without CKD and T2DM with CKD (hemodialysis).

Materials and Methods: The study is a comparative prospective case-controlled study that was conducted on 90 individuals, comprised of thirty patients who had T_2DM without CKD and thirty patients who had T_2DM with CKD (hemodialysis) and 30 healthy subjects who participated as control group. The levels of visfatin was measured by ELISA technique whereas the levels of urea, creatinine, potassium and sodium were measured by colorimetric method according to the manufacturer manual.

Result: The age of participants was range from (35-65) years, BMI range (19.115-32.281), Mean BMI of participants cases group in T_2DM without CKD (31.555 ± 1.570), T_2DM with CKD (Hemodialysis) state (19.840 ± 2.086), this study found that the serum concentration of Visfatin was significantly higher in patients than controls (P value 0.00004). On the other hand this study found (Urea, Creatinine, Potassium, Sodium) were a statistically significant ($p \leq 0.05$) in patients compared to controls (P value 0.00008, 0.000003, 0.00009, 0.0008) respectively but the result was normal concentration in T_2DM without CKD.

Conclusion: The conclusion was the increased level of visfatin in all groups of patients, the rise was gradually from T_2DM without CKD to T_2DM with CKD (hemodialysis) state. The Visfatin could be served as a diagnostic marker in T_2DM patients to predict the possibility to develop nephropathy High level of potassium in T_2DM with hemodialysis, but Na was low in same group, The overweight T_2DM without CKD Possible cause of the development of diabetic nephropathy in the future, The present study found the urea, creatinine were normal concentration in patients T_2DM without CKD.

Key Wards: Type 2 diabetic Mellitus, Hemodialysis**,** Visfatin.

Introduction

The World Health Organization estimated that globally 422 million adults were living with diabetes in 2014, Diabetes has become a major epidemic worldwide. Type 2 diabetes mellitus accounts for 90–95% of all diabetes. Disease develops insidiously through periods of increased insulin secretion, insulin resistance, impaired glucose tolerance, and β-cell dysfunction[1].on the basis of laboratory findings, as a fasting venous plasma glucose concentration of 7.0 mmol/L or more (on more than one occasion or once in the presence of diabetes symptoms) or a random venous plasma glucose concentration of 11.1 mmol/L or more[2,3].Patients have minimal symptoms, are not prone to ketosis, and are not dependent on insulin to prevent ketonuria ,Most patients acquire the disease after age 40, but it occur in younger people[4].Chronic kidney disease (CKD) is a frequent complication of type 2 diabetes mellitus (T2DM), CKD is a major global health burden because of its high prevalence and a potent risk factor for kidney failure, cardiovascular events, and early death[5]. In 2016, nearly 125,000 people in the United States started treatment for ESKD, and more than 726,000 (2 in every 1,000 people) were on dialysis or were living with a kidney transplant[6].CKD, is defined as a progressive loss of kidney function occurring over several months to years; it is characterized by the gradual replacement of normal kidney structure with fibrotic tissues. When these structural changes become conspicuous, it results in decreased kidneys' ability to process waste in the blood and perform other functions. During early stages patients may present with normal or slight decrease in Glomerular filtration rate (GFR) and Albuminuria; later it

progresses, leading to end stage renal disease (ESRD) or kidney failure. ESRD is irreversible and fatal, unless treated by dialysis or kidney transplant[7].

Visfatin was first described in 2005 by Fukuhara et al.[8]. They showed that visfatin is expressed mainly by visceral adipose tissue with insulin-like effects. Visfatin, also known as pre-B-cell colony enhancing factor (PBEF), has been described as an adipokine with a potential glucose-lowering effect due to its nicotinamide phosphoribosyl transferase (NAMPT) activity[9]. Visfatin is a 52-kDa adipocytokine that is predominantly secreted by visceral adipose tissue. It is also found in skeletal muscle, liver, bone marrow, lymphocytes and placenta. It has insulinomimetic effects by activating the insulin signal transduction pathway through binding to the same receptor. Systemic visfatin concentrations are acutely regulated by glucose and insulin. In vitro exposure of adipocytes to glucose and human studies of circulating visfatin both showed an increase in visfatin concentration [10]. Biomarker studies showed that the circulating level of visfatin are upregulated in T_2DM[11].

Materials and Methods

A case control, hospital based study, the protocol of this study was approved by the scientific committee of Tikrit University-College of Medicine, and the agreement of the attendance to Al-Hayat dialysis center to collect the sample from the patients was approved by the al-karama teaching hospital. This study was carried out at the Al-Hayat dialysis center in Baghdad City- Iraq from the 1st of February 2020 to the end of May 2020. A verbal consent was taken from each patient included in this study whether considered as a case or control. This study conducted on (90) participants, The study comprised of three groups, first group 30 adult patients who underwent Kidney dialysis unit as CKD patients with type 2 diabetic (on hemodialysis). Second group 30 adult patients who have type 2 diabetic, their ages were between 35 to 65 years, while 30 adult persons who look healthy with no prior medical or family history of CKD and type 2 diabetic as a control group participated in this study, their ages were from 35 to 65 year. By using a sterile disposable syringe 5 mls of venous blood sample was drawn from each patients included in this study at the morning and was kept in a plain tube and allowed to clot at room temperature, then each sample was centrifuged at 6000 rpm to obtain serum. The serum was aspirated then divided into aliquots in plastic tubes and stored at -20 ^{0}C until the time of estimation.

Serum of the patients and controls had assay: Visfatin by ELISA and Urea, creatinine sodium and potassium by photometric manufacture

Results

BMI range (19.115-32.281) they are statistically significant P-value 0.00006, BMI for cases group in T_2DM with CKD (hemodialysis) state (19.840 ± 2.086) ,T_2DM without CKD (hemodialysis) state (31.555 ±1.570) and control group (23.387±2.274), P-Value (0.00006) These statistics were summarized in **(table.1)**.

Table (1): Descriptive characteristics of BMI between studied groups

BMI*	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	19.840	31.555	23.387
SD	2.086	1.570	2.274
95% CI	(19.115, 20.565)	(30.830, 32.281)	(22.662, 24.112)

This study also shows that the mean serum level of Visfatin was higher in CKD patients with type 2 diabetic (on hemodialysis)(5.991± 1.194 ng/ml) , type 2 diabetic without CKD(3.023 ± 0.4956 ng/ml) as compared with the control group (0.779 ± 0.1439 ng/ml). This result was highly significant at a P value of 0.00004, see the **(table.2)**.

Table (2): Descriptive characteristics of serum Visfatin ng/mL level between studied groups

Visfatin ng/mL	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	5.991	3.023	0.779
SD	1.194	0.4956	0.1439
95% CI	(5.719, 6.264)	(2.7505, 3.2955)	(0.5061, 1.0512)

This study also shows that the mean serum level of Urea was higher in CKD patients with type 2 diabetic (on hemodialysis) (132.660±43.54mg/Dl), type 2 diabetic without CKD (25.003 ± 3.213 mg/Dl) as compared with the control group (21.433 ± 2.060 mg/Dl). This result was highly significant at a P value of 0.00008, see the **(table.3)**.

Table (3): Descriptive characteristics of Blood Urea mg/dL levels between studied groups

Urea mg/dL	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	132.660	25.003	21.433
SD	43.54	3.213	2.060
95% CI	(123.50, 141.82)	(15.847, 34.160)	(12.277, 30.590)

This study also shows that the mean serum level of creatinine was higher in CKD patients with type 2 diabetic (on hemodialysis) (5.822 ± 1.767 mg/Dl), type 2 diabetic without CKD (0.540 ± 0.0851mg/Dl) as compared with the control group (0.540 ± 0.0851 mg/Dl). This result was highly significant at a P value of 0.000003, see the **(table.4)**.

Table (4): Descriptive characteristics of serum Creatinine mg/dL levels between studied groups

Creatinine mg/dL	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	5.822	0.458	0.540
SD	1.767	0.0803	0.0851
95% CI	(5.451, 6.193)	(0.0873, 0.8294)	(0.1687, 0.9107)

This study also shows that the mean serum level of Potassium was higher in CKD patients with type 2 diabetic (on hemodialysis) (5.9050 ± 0.5059 mmol/L), type 2 diabetic without CKD (4.2957 ± 0.3881 mmol/L) as compared with the control group (4.0733 ± 0.2935 mmol/L). This result was highly significant at a P value of 0.00009, see the **(table 5)**.

Table (5): Descriptive characteristics of Potassium mmol/L between studied groups

Potassium mmol/L	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	5.9050	4.2957	4.0733
SD	0.5059	0.3881	0.2935
95% CI	(5.7579, 6.0521)	(4.1486, 4.4427)	(3.9263, 4.2204)

This study also shows that the mean serum level of sodium was higher in CKD patients with type 2 diabetic (on hemodialysis) (131.136 ± 3.268 mmol/L), type 2 diabetic without CKD (138.047 ± 3.429mmol/L) as compared with the control group (139.233 ± 1.794mmol/L). This result was highly significant at a P value of 0.0008, see the **(table.6)**.

Table (6): Descriptive characteristics of Na mmol/L between studied groups

Na mmol/L	T_2DM with hemodialysis (n=30)	T_2DM without hemodialysis (n=30)	Control (n=30)
Mean	131.136	138.047	139.233
SD	3.268	3.429	1.794
95% CI	(130.075, 132.197)	(136.985, 139.108)	(138.17,140.29)

Regarding positive correlation between fasting blood glucose and Visfatin in hemodialysis state, (P value = 0.000) as summarized in **(Figure)**.

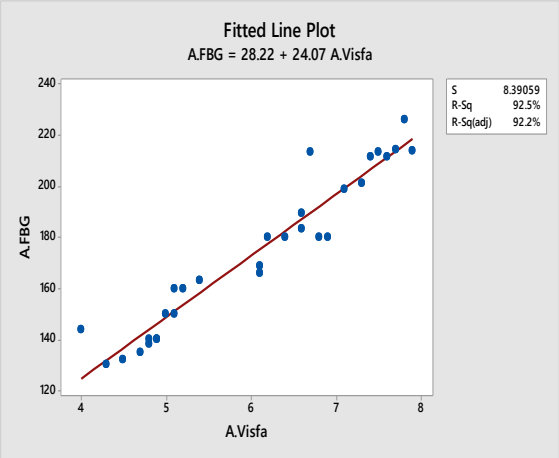

Figure: Correlation between FBG and Visfatin (ng/ml) in hemodialysis state

Discussion

This study was found of the patients levels was Visfatin level was significantly higher in all studied groups state than in controls, which is compatible with *Mageswari R et al ,2019 and Tabur S et al ,2016* whose found a highly significant difference in Visfatin levels between of patients and controls and can investigated the role of visfatin in early diabetic nephropathy [12,(13] ,the current study agreement with *Kacso AC et al ,2016.* who found a highly significant difference in visfatin levels between of patients and controls[14],In the current study, the level of visfatin showed to increase significantly in the T_2DM in comparison with the control and significantly increase in the hemodialysis patients with T_2DM in a comparison with both T_2DM and control groups, which may indicate early predict to diabetic complications such chronic kidney diseases. Pearson's correlation have shown significant positive correlation between visfatin and FBS level hemodialysis state. The biological mechanisms involving visfatin in the pathogenesis of T_2DM are not well understood. Visfatin as an adipokine has recently been identified and named as such because of its much greater expression in visceral fat than in subcutaneous adipose tissue. In keeping with its insulin-mimetic effects, visfatin was as effective as insulin in reducing hyperglycemia in insulin-deficient diabetic mice. Visfatin was also bound to and activated insulin receptors, causing receptor phosphorylation and the activation of the downstream signaling molecules[15]

Results obtained in this study revealed that urea level in the serum of patients in hemodialysis group (before hemodialysis) was significantly higher than that of T_2DM without hemodialysis and control groups. This finding is in agreement with that of *Gotta V, Marsenic O et al ,2020*[16] and *Obi Y, Rhee CM et al , 2016* [17].

In the current study, the creatinine was significantly higher in T_2DM with hemodialysis state when compared to T_2DM without hemodialysis and controls. This association between Creatinine and diabetic CKD was described by several studies including that by *Norris KC et al. ,2018* [18] and *Nowak N. ,2018* [19]

The progression of kidney damage is marked by the rise in two important chemical substances in the blood, creatinine and urea whose evaluation in serum helps to assess GFR followed by renal function. However, creatinine nor urea is directly toxic and they are only a measure of kidney function[20]**.**

The level of potassium was significantly higher in patients subjected to this study in group of patients with diabetic hemodialysis when compared to control .Similarly, a study by ***Dhondup T et al, 2017*** [21].Found a normal concentration for our results of potassium in T_2DM without CKD patients but more than concentration of control but it is insignificant , Similarly ,a study by ***Rajagambeeram R et al ,2020*** [22],Who found the serum levels are not significantly altered.

Hyperglycemia leads to hyperosmolarity , this in turn to dehydration of cells, thus causing an increase in potassium extrusion from cells into ECF[23]. Hyperkalemia is one of the common electrolyte disorders in patients with CKD. Decreased glomerular filtration and tubular potassium secretion, often coupled with a generous dietary potassium intake, are the major causes[24].

Sodium were found to be lower in patients' with T_2DM with hemodialysis when compared to control, This association between Na and Diabetic kidney disease was described by several studies including that by ***Lin J et al. ,2018*** [25] and ***Pirklbauer M et al ,2020*** [26].

Hyponatremia and volume overload in dialysis patients are associated with elevated mortality. The major causes of pre-dialysis hyponatremia are insufficient water removal during dialysis and increased oral water intake. Volume overload is the result of total body Na+ excess[25].

In our study found a normal concentration for our results of sodium in T_2DM without CKD patients.

The study showed that the BMI in T_2DM without hemodialysis lie within scores 30-32, it is highly significant comparable with diabetic CKD and control. Moreover, ***Omar MS et al,2016*** concluded that the Obesity is a major risk factor for many non-communicable disease (NCDs) and its complications, including T_2DM, Cardiovascular disease (CVD), hypertension, and stroke[27].The percentage of patients with BMI more than 30 was 74.3%, and if added to overweight we come up with a percentage of 95.5%. The average BMI reported was 33.54 ± 5.94 kg/m2.These results highlight the need for lifestyle interventions, education for obesity and overweight[28].

In the present study showed that the BMI in T_2DM with hemodialysis it is significantly lower comparable with diabetic without CKD and control , compatible with Medeiros MC et al, 2020 and concluded that the determined the prevalence of sarcopenia in patients undergoing hemodialysis and evaluated the association in these patients of diabetes [29] .Sarcopenia can be defined as a gradual and generalized loss of muscular mass and muscular strength, which may be caused by advanced age, chronic diseases, physical inactivity, use of some medicines, and/or nutritional deficit. As a consequence of protein loss and metabolic damage caused by uremia, chronic kidney disease can result in reduced muscle synthesis and sarcopenia and is found to be more prevalent in dialysis patients (37.0% in men and 29.3% in women). Diabetes mellitus (DM) is one of the main causes of chronic kidney disease (CKD) and is also associated with increased risk of sarcopenia, which may be related to peripheral neuropathy due to reduced neuronal stimulation, insulin resistance, a pro-inflammatory condition, mitochondrial dysfunction, and oxidative injury[30].

Conclusion

The increased level of visfatin in all groups of patients, the rise was gradually from T_2DM without CKD to T_2DM with CKD (hemodialysis) state. The Visfatin could be served as a diagnostic marker in T2DM patients to predict the possibility to develop nephropathy, High level of potassium in T_2DM with hemodialysis, but Na was low in same group, The overweight T_2DM without CKD Possible cause of the development of diabetic nephropathy in the future, The present study found the urea, creatinine were normal concentration in patients T_2DM without CKD.

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دراسة دور فسفاتين في مصل دم المصابين بداء السكري من النوع الثاني مع وبدون غسيل الكلى

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الملخص

الخلفية : نمت حدوث وانتشار مرض السكري بشكل كبير في جميع أنحاء العالم، ويرجع ذلك أساسا إلى الزيادة في مرض السكري النوع الثاني. مرض الكلى المزمن هو مضاعفات متكرر لمرض السكري من النوع الثاني ، للفسفاتين له تأثيرات محاكية للأنسولين من خلال تنشيط مسار تحويل إشارة الأنسولين من خلال الارتباط بنفس المستقبلات. وفقًا لدراستنا ، فقد أظهرت العلاقة بين فسفاتين ، علامة بيولوجية محددة لـ لـ مرض السكري من النوع الثاني بدون مرض الكلوي مزمن و مرض السكري من النوع الثاني مع مرض الكلوي مزمن (غسيل الكلى).

المواد وطرق العمل: لدراسة عبارة عن دراسة مقارنة مستقبلية مضبوطة بالحالة تم إجراؤها على 90 فردًا ، تتألف من ثلاثين مريضًا لـ مرض السكري من النوع الثاني بدون مرض الكلوي مزمن و ثلاثون مريضا لديهم مرض السكري من النوع الثاني مع مرض الكلوي مزمن (غسيل الكلى)و 30 من شخصًا أصحاء شاركوا كمجموعة ضابطة.. تم قياس مستويات فسفاتين بتقنية الايلايزا بينما تم قياس مستويات اليوريا والكرياتينين والبوتاسيوم والصوديوم بطريقة القياس اللوني حسب دليل الشركة المصنعة.

النتائج: تراوح عمر المشاركين من (35–65) سنة ، نطاق مؤشر كتلة الجسم (19.115–32.281) ، متوسط مؤشر كتلة الجسم للمشاركين في مجموعة السكري من النوع الثاني بدون فشل الكلوي (31.55±1,57) ،مرضى السكري من النوع الثاني مع فشل الكلوي قبل الغسل الكلية (19.84±2,06)، وجدت هذه الدراسة أن تركيز مصل تركيز الفسفاتين كان اكثر في المرضى مقارنه مع مجموعه المقارنة (القيمة المعنوية = 0,00004). من ناحية أخرى، وجدت هذه الدراسة أن (اليوريا، الكرياتينين، البوتاسيوم، الصوديوم) وجود زيادة ذات قيمة معنوية (اقل من 0,05) عند مقارنة بمجاميع المقارنة القيمة المعنوية = (0,00008 ، 0,00003 ، 0,00009 ، 0,00008) على التوالي ولكن كانت النتائج طبيعية في مجموعه مرضى السكري من النوع الثاني من دون فشل الكلوي.

الاستنتاج الاستنتاج هو زيادة مستوى فسفاتين في جميع مجموعات المرضى ، وكان الارتفاع تدريجياً من مجموعة السكري من النوع الثاني بدون فشل الكلوي إلى مرض السكري من النوع الثاني مع مرض الكلوي مزمن (غسيل الكلى). يمكن استخدام فسفاتين كعلامة تشخيصية في مرضى السكري من النوع الثاني للتنبؤ بإمكانية الإصابة باعتلال الكلية. ارتفاع مستوى البوتاسيوم في مجموعه السكري من النوع الثاني مع غسيل الكلى ، ولكن الصوديوم كان منخفضًا في نفس المجموعة ، والوزن الزائد البوتاسيوم في مجموعه السكري من النوع الثاني دون غسيل الكلى هو السبب المحتمل لتطور اعتلال الكلية السكري في المستقبل ، وجدت الدراسة الحالية أن اليوريا والكرياتينين كان تركيزًا طبيعيًا في المرضى البوتاسيوم في مجموعه السكري من النوع الثاني دون غسيل الكلى .